activities; and threatening the survival of patients if no treatment is performed for protecting nerve cells or neurons at risk in the lesion site as early as possible. Consequently, the treatment of cerebral apoplexy should be initiated without a moment's delay. Even the period of time during the CT inspection of the brain is, to put it strongly, a factor for reducing the possibility of recovery for patients with cerebral apoplexy. Surely, the treatment of acute cerebral apoplexy is a struggle not only against the cerebral apoplectic lesion but also against the time after its onset. Quite unfortunately, at present, whatever the disease type of cerebral apoplexy is (cerebral infarction, cerebral thrombosis, cerebral embolism, cerebral hemorrhage, subarachnoidal hemorrhage or transient ischemic attack), it is a fact that there are few known drugs showing potent effects, even if they are administered immediately after the onset of cerebral apoplexy.

The important thing in the treatment of cerebral apoplexy is not always related to the acute stages of the disease. Even if, as a result of administration of some potent neuroprotective agents, necrosis or apoptosis-like cell death of brain cells (including glial cells) or nerve cells can be temporarily prevented; and unless the regeneration and/or the reconstruction of the brain blood vessels in the lesion site can occur subsequently, the brain cells and the nerve cells (neurons) in the same site are likely to degenerate over a long

time period. However, at present, almost no drugs for stimulating the regeneration and/or the reconstruction of the cerebral blood vessels are known. These facts can be explained more concretely by using the example of a cerebral infarct lesion caused by permanent occlusion of the cortical branch of the unilateral middle cerebral artery (MCA). When the MCA is permanently occluded, nerve cells or neurons in the site, to which nutrition is supplied only through the MCA (i.e. the nerve cells or neurons in the ischemic core) fall into necrosis and form a cerebral infarct lesion unless the MCA is soon recanalized and reperfused. Consequently, no drugs can rescue the brain tissue in the ischemic core. As previously described in the specifications of the JP98/365560 and PCT/JP/02550 (Brain cell or nerve cell-protective agents comprising ginsenoside Rb_1), and in the present specification, gradually progressing nerve cell death, which is different from necrosis, is defined as "apoptosis of nerve cells" or "apoptosis-like nerve cell death".

In the ischemic penumbra, since the supply of the blood flow from the MCA is terminated and the vascular networks in that lesion are extremely decreased; and since the blood supply continues, at least in part, from the cortical branches of the anterior cerebral arteries and the posterior cerebral arteries, the nerve cells or neurons in the ischemic penumbra may survive for a short period after permanent MCA occlusion in a critical condition. It is well known that, if no measures are taken,

apoptosis-like nerve cell (neuron) death gradually develops in the ischemic penumbra, and the original lesion (ischemic penumbra) is totally and drastically changed to a cerebral infarct lesion. From the clinical standpoint, to rescue the nerve cells or neurons in the original ischemic penumbra is the most important thing to do. However, even if the nerve cells in that ischemic penumbra lesion can survive temporarily as a result of applying potent neuroprotective agents as described above, unless the vascular networks in that lesion, which are disrupted or decreased due to the permanent MCA occlusion, can be regenerated and/or reconstructed, the nerve cells in that lesion may eventually die. Consequently, with regard to the conditions required for neuroprotective agents, stimulation of the regeneration and/or the reconstruction of the disrupted vascular networks in the ischemic penumbra is essential in addition to a direct neuroprotective action.

A further problem in the treatment of cerebral apoplexy is the histological features or characteristics of the brain. Since each region in the brain constitutes complex information networks with the others through synaptic interactions, if one region is damaged, the damage frequently proceeds later in a staggered sequence to the other related regions connected synaptically with the original region (sometimes synaptic connection is designated as fiber connection). For example, it is reported that when a cerebral infarct lesion (the primary